

A CONSPECTUS OF BACTERIAL DISEASES OF PLANTS

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All our knowledge of these diseases has come within a generation. It began thirty-six years ago with the announcement of the bacterial origin of pear blight by Professor T. J. Burrill of the University of Illinois, who is with us to-day. During the first half of that period progress was slow and doubt universal, especially in Europe.

It is now eighteen years since I ventured the statement,¹ that "there are in all probability as many bacterial diseases of plants as of animals." This statement was received with much skepticism, not to mention active opposition, but time has more than borne out my statement, and there is now no one left to dispute it. To-day I will venture another, and broader generalization, to wit: It appears likely that eventually a bacterial disease will be found in every family of plants, from lowest to highest. This prediction is based on the fact that although the field is still a very new one, with no workers in most parts of the world, such diseases have been reported from every continent, and are already known to occur in plants of one hundred and forty genera distributed through more than fifty families.

DISTRIBUTION

Following Engler's arrangement, I will list these families that you may see how wide is the distribution of bacterial diseases in plants and how utterly wrong were those who said that there were no such diseases, and also those who conceded a little but said that they were very rare and restricted to the soft underground parts of a few bulbous and tuberous plants, and generally preceded by fungi. In this list, I have included only the flowering plants, but some of the cryptogams are also

¹ *Am. Nat.* 30: p. 627. 1896.

subject to bacterial attack. The number following the family name indicates the number of bacterial diseases known within the limits of the family. The total of the figures, however, will not give the number of bacterial parasites, because some of the diseases overlap.

TABLE I

SHOWING THE FAMILIES OF FLOWERING PLANTS ARRANGED SERIALLY FROM LOWEST TO HIGHEST. THOSE CONTAINING GENERA SUBJECT TO BACTERIAL DISEASES ARE UNDERSCORED, AND WHEN SEVERAL DISEASES HAVE BEEN RECOGNIZED THEIR NUMBER IS ALSO GIVEN

1. <u>Cycadaceae</u>	34. <u>Juncaceae</u>	68. <u>Myzodendraceae</u>
2. <u>Ginkgoaceae</u>	35. <u>Stemonaceae</u>	69. <u>Santalaceae</u>
3. <u>Taxaceae</u>	36. <u>Melanthiaceae</u>	70. <u>Grubbiaceae</u>
4. <u>Pinaceae</u> 2	37. <u>Liliaceae</u> 3	71. <u>Opiliaceae</u>
5. <u>Gnetaceae</u>	38. <u>Convallariaceae</u>	72. <u>Olacaceae</u>
6. <u>Typhaceae</u>	39. <u>Smilacaceae</u>	73. <u>Balanophoraceae</u>
7. <u>Pandanaceae</u>	36. }	74. <u>Aristolochiaceae</u>
8. <u>Sparganiaceae</u>	37. } <i>Liliaceae</i>	75. <u>Rafflesiaceae</u>
9. <u>Potamogetonaceae</u>	38. }	76. <u>Hydnoraceae</u>
10. <u>Naiadaceae</u>	39. }	77. <u>Polygonaceae</u> 2
11. <u>Aponogetonaceae</u>	40. <u>Haemodoraceae</u>	78. <u>Chenopodiaceae</u> 4
12. <u>Scheuchzeriaceae</u>	41. <u>Amaryllidaceae</u>	79. <u>Amaranthaceae</u>
12. <u>Juncaginaceae</u>	42. <u>Velloziaceae</u>	80. <u>Nyctaginaceae</u>
13. <u>Alismaceae</u>	43. <u>Taccaceae</u>	81. <u>Batidaceae</u>
14. <u>Butomaceae</u>	44. <u>Dioscoreaceae</u>	82. <u>Theligonaceae</u>
15. <u>Vallisneriaceae</u>	45. <u>Iridaceae</u>	82. <u>Cynocrambaceae</u>
15. <u>Hydrocharitaceae</u>	46. <u>Musaceae</u>	83. <u>Phytolaccaceae</u>
16. <u>Triuridaceae</u>	47. <u>Zingiberaceae</u>	84. <u>Aizoaceae</u>
17. <u>Poaceae</u>	48. <u>Cannaceae</u>	85. <u>Portulacaceae</u>
17. <u>Gramineae</u> 7	49. <u>Marantaceae</u>	86. <u>Basellaceae</u>
18. <u>Cyperaceae</u>	50. <u>Burmanniaceae</u>	87. <u>Silenaceae</u>
19. <u>Phoenicaceae</u>	51. <u>Orchidaceae</u>	87. <u>Caryophyllaceae</u> 2
19. <u>Palmae</u>	52. <u>Casuarinaceae</u>	88. <u>Nymphaeaceae</u>
20. <u>Cyclanthaceae</u>	53. <u>Saururaceae</u>	89. <u>Ceratophyllaceae</u>
21. <u>Araceae</u>	54. <u>Piperaceae</u>	90. <u>Trochodendraceae</u>
22. <u>Lemnaceae</u>	55. <u>Chloranthaceae</u>	91. <u>Ranunculaceae</u>
23. <u>Flagellariaceae</u>	56. <u>Salicaceae</u> 2	92. <u>Lardizabalaceae</u>
24. <u>Baloskionaceae</u>	57. <u>Myricaceae</u>	93. <u>Berberidaceae</u>
24. <u>Restionaceae</u>	58. <u>Balanopsidaceae</u>	94. <u>Menispermaceae</u>
25. <u>Centrolepidaceae</u>	59. <u>Leitneriaceae</u>	95. <u>Magnoliaceae</u>
26. <u>Mayacaceae</u>	60. <u>Juglandaceae</u> 2	96. <u>Calycanthaceae</u>
27. <u>Xyridaceae</u>	61. <u>Betulaceae</u>	97. <u>Lactoridaceae</u>
28. <u>Eriocaulaceae</u>	62. <u>Fagaceae</u>	98. <u>Annonaceae</u>
29. <u>Rapateaceae</u>	63. <u>Ulmaceae</u>	99. <u>Myristicaceae</u>
30. <u>Bromeliaceae</u>	64. <u>Moraceae</u>	100. <u>Gomortegaceae</u>
31. <u>Commelinaceae</u>	65. <u>Urticaceae</u> 4	101. <u>Monimiaceae</u>
32. <u>Pontederiaceae</u>	66. <u>Proteaceae</u>	102. <u>Lauraceae</u>
33. <u>Philydraceae</u>	67. <u>Loranthaceae</u>	103. <u>Hernandiaceae</u>

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|------------------------------|------------------------------|-------------------------------|
| 104. <u>Papaveraceae</u> | 140. <u>Tropaeolaceae</u> 3 | 184. <u>Malvaceae</u> 2 |
| 105. <u>Brassicaceae</u> | 141. <u>Linaceae</u> | 185. <u>Triplachitonaceae</u> |
| 105. <u>Cruciferae</u> 5 | 142. <u>Humiriaceae</u> | 186. <u>Bombacaceae</u> |
| 106. <u>Tovariaceae</u> | 143. <u>Erythroxylaceae</u> | 187. <u>Sterculiaceae</u> |
| 107. <u>Capparidaceae</u> | 144. <u>Zygophyllaceae</u> | 188. <u>Scytopetalaceae</u> |
| 108. <u>Resedaceae</u> | 145. <u>Cneoraceae</u> | 189. <u>Dilleniaceae</u> |
| 109. <u>Moringaceae</u> | 146. <u>Rutaceae</u> | 190. <u>Eucryphiaceae</u> |
| 110. <u>Sarraceniaceae</u> | 147. <u>Simaroubaceae</u> | 191. <u>Ochnaceae</u> |
| 111. <u>Nepenthaceae</u> | 148. <u>Balsameaceae</u> | 192. <u>Caryocaraceae</u> |
| 112. <u>Droseraceae</u> | 148. <u>Burseraceae</u> | 193. <u>Marcgraviaceae</u> |
| 113. <u>Podostemonaceae</u> | 149. <u>Meliaceae</u> | 194. <u>Quiinaceae</u> |
| 114. <u>Hydrostachyaceae</u> | 150. <u>Malpighiaceae</u> | 195. <u>Theaceae</u> |
| 115. <u>Crassulaceae</u> | 151. <u>Trigoniaceae</u> | 196. <u>Hypericaceae</u> |
| 116. <u>Penthoraceae</u> | 152. <u>Vochyaceae</u> | 197. <u>Clusiaceae</u> |
| 115. } <u>Crassulaceae</u> | 152. <u>Vochysiaceae</u> | 196. } <u>Guttiferae</u> |
| 116. } | 153. <u>Tremandraceae</u> | 197. } |
| 117. <u>Cephalotaceae</u> | 154. <u>Polygalaceae</u> | 198. <u>Dipterocarpaceae</u> |
| 118. <u>Saxifragaceae</u> | 155. <u>Dichapetalaceae</u> | 199. <u>Elatinaceae</u> |
| 119. <u>Hydrangeaceae</u> | 156. <u>Euphorbiaceae</u> | 200. <u>Frankeniaceae</u> |
| 120. <u>Escalloniaceae</u> | 157. <u>Callitrichaceae</u> | 201. <u>Tamaricaceae</u> |
| 121. <u>Grossulariaceae</u> | 158. <u>Buxaceae</u> | 202. <u>Fouquieriaceae</u> |
| 118. } | 159. <u>Coriariaceae</u> | 203. <u>Cistaceae</u> |
| 119. } <u>Saxifragaceae</u> | 160. <u>Empetraceae</u> | 204. <u>Bixaceae</u> |
| 120. } | 161. <u>Limnanthaceae</u> | 205. <u>Cochlospermaceae</u> |
| 121. } | 162. <u>Anacardiaceae</u> | 206. <u>Koeberliniaceae</u> |
| 122. <u>Pittosporaceae</u> | 163. <u>Cyrillaceae</u> | 207. <u>Canellaceae</u> |
| 123. <u>Brunelliaceae</u> | 164. <u>Pentaphylacaceae</u> | 208. <u>Violaceae</u> |
| 124. <u>Cunoniaceae</u> | 165. <u>Corynocarpaceae</u> | 209. <u>Flacourtiaceae</u> |
| 125. <u>Myrothamnaceae</u> | 166. <u>Aquifoliaceae</u> | 210. <u>Stachyuraceae</u> |
| 126. <u>Bruniaceae</u> | 167. <u>Celastraceae</u> | 211. <u>Turneraceae</u> |
| 127. <u>Hamamelidaceae</u> | 168. <u>Hippocrateaceae</u> | 212. <u>Malesherbiaceae</u> |
| 128. <u>Platanaceae</u> | 169. <u>Stackhousiaceae</u> | 213. <u>Passifloraceae</u> |
| 129. <u>Crossosomataceae</u> | 170. <u>Staphyleaceae</u> | 214. <u>Achariaceae</u> |
| 130. <u>Rosaceae</u> | 171. <u>Icacinaceae</u> | 215. <u>Papayaceae</u> |
| 131. <u>Malaceae</u> | 172. <u>Aceraceae</u> | 215. <u>Caricaceae</u> |
| 132. <u>Amygdalaceae</u> | 173. <u>Aesculaceae</u> | 216. <u>Loasaceae</u> |
| 130. } | 173. <u>Hippocastanaceae</u> | 217. <u>Datisceae</u> |
| 131. } <u>Rosaceae</u> 6 | 174. <u>Sapindaceae</u> | 218. <u>Begoniaceae</u> |
| 132. } | 175. <u>Sabiaceae</u> | 219. <u>Ancistrocladaceae</u> |
| 133. <u>Connaraceae</u> | 176. <u>Bersamaceae</u> | 220. <u>Cactaceae</u> |
| 134. <u>Mimosaceae</u> | 176. <u>Melanthaceae</u> | 221. <u>Geissolomaceae</u> |
| 135. <u>Caesalpinaceae</u> | 177. <u>Impatiaceae</u> | 222. <u>Penaeaceae</u> |
| 136. <u>Krameriaceae</u> | 177. <u>Balsaminaceae</u> | 223. <u>Oliniaceae</u> |
| 137. <u>Fabaceae</u> | 178. <u>Rhamnaceae</u> | 224. <u>Thymelaeaceae</u> |
| 134. } | 179. <u>Vitaceae</u> 3 | 225. <u>Elaeagnaceae</u> |
| 135. } <u>Leguminosae</u> 5 | 180. <u>Elaeocarpaceae</u> | 226. <u>Lythraceae</u> |
| 136. } | 181. <u>Schizolaenaceae</u> | 227. <u>Blattiaceae</u> |
| 137. } | 181. <u>Chlaenaceae</u> | 227. <u>Sonneratiaceae</u> |
| 138. <u>Geraniaceae</u> 2 | 182. <u>Gonystylaceae</u> | 228. <u>Crypteroniaceae</u> |
| 139. <u>Oxalidaceae</u> | 183. <u>Tiliaceae</u> | 229. <u>Punicaceae</u> |

230. <u>Lecythidaceae</u>	252. <u>Primulaceae</u>	275. <u>Bignoniaceae</u>
231. <u>Rhizophoraceae</u>	253. <u>Plumbaginaceae</u>	276. <u>Pedaliaceae</u>
232. <u>Combretaceae</u>	254. <u>Sapotaceae</u>	277. <u>Martyniaceae</u>
233. <u>Myrtaceae</u>	255. <u>Diospyraceae</u>	278. <u>Orobanchaceae</u>
234. <u>Melastomataceae</u>	255. <u>Ebenaceae</u>	279. <u>Gesneriaceae</u>
235. <u>Onagraceae</u>	256. <u>Styracaceae</u>	280. <u>Columelliaceae</u>
236. <u>Trapaceae</u>	257. <u>Symplocaceae</u>	281. <u>Pinguiculaceae</u>
236. <u>Hydrocaryaceae</u>	258. <u>Oleaceae</u> 2	281. <u>Lentibulariaceae</u>
237. <u>Haloragidaceae</u>	259. <u>Salvadoraceae</u>	282. <u>Globulariaceae</u>
237. <u>Halorrhagidaceae</u>	260. <u>Loganiaceae</u>	283. <u>Acanthaceae</u>
238. <u>Cynomoriaceae</u>	261. <u>Gentianaceae</u>	284. <u>Myoporaceae</u>
239. <u>Araliaceae</u> 2	262. <u>Menyanthaceae</u>	285. <u>Phrymaceae</u>
240. <u>Apiaceae</u>	261. } <u>Gentianaceae</u>	286. <u>Plantaginaceae</u>
240. <u>Umbelliferae</u> 3	262. }	287. <u>Rubiaceae</u>
241. <u>Cornaceae</u>	263. <u>Apocynaceae</u>	288. <u>Caprifoliaceae</u>
242. <u>Clethraceae</u>	264. <u>Asclepiadaceae</u>	289. <u>Adoxaceae</u>
243. <u>Pyrolaceae</u>	265. <u>Convolvulaceae</u>	290. <u>Valerianaceae</u>
244. <u>Monotropaceae</u>	266. <u>Cuscutaceae</u>	291. <u>Dipsacaceae</u>
243. } <u>Pyrolaceae</u>	265. } <u>Convolvulaceae</u>	292. <u>Cucurbitaceae</u> 3
244. }	266. }	293. <u>Campanulaceae</u>
245. <u>Lennoaceae</u>	267. <u>Polemoniaceae</u>	294. <u>Goodeniaceae</u>
246. <u>Ericaceae</u>	268. <u>Hydrophyllaceae</u>	295. <u>Candolleaceae</u>
247. <u>Vacciniaceae</u>	269. <u>Boraginaceae</u>	296. <u>Calyceraceae</u>
246. } <u>Ericaceae</u>	270. <u>Verbenaceae</u>	297. <u>Cichoriaceae</u>
247. }	271. <u>Menthaceae</u>	298. <u>Ambrosiaceae</u>
248. <u>Epacridaceae</u>	271. <u>Labiatae</u>	299. <u>Asteraceae</u>
249. <u>Diapensiaceae</u>	272. <u>Nolanaceae</u>	297. } <u>Compositae</u> 3
250. <u>Theophrastaceae</u>	273. <u>Solanaceae</u> 9	298. }
251. <u>Myrsinaceae</u>	274. <u>Scrophulariaceae</u>	299. }

The widest gap, it will be observed, is between *Cruciferae* and *Rosaceae*, but I believe this represents nothing more than lack of knowledge.

Also I should like to list the genera within the limits of which one or more species are now said to be subject to attack, because many of these genera contain plants of great economic importance. Where I have some personal knowledge of the subject I have italicized the genus name, and in what follows the reader will naturally expect me to draw illustrations principally from the diseases most familiar to me.

TABLE II

SHOWING GENERA OF FLOWERING PLANTS SUBJECT TO DISEASES OF BACTERIAL ORIGIN

<i>Macrozamia</i>	<i>Bromus</i>	<i>Avena</i>	<i>Phleum</i>
<i>Pinus</i>	<i>Zea</i>	<i>Saccharum</i>	<i>Poa</i>
<i>Dactylis</i>	<i>Andropogon</i>	<i>Triticum</i>	<i>Cocos</i>

<i>Oreodoxa</i>	<i>Beta</i>	<i>Prosopis</i> (?)	<i>Syringa</i>
<i>Richardia</i>	<i>Amaranthus</i>	<i>Erythrina</i>	<i>Olea</i>
<i>Amorphophallus</i>	<i>Dianthus</i>	<i>Geranium</i>	<i>Fraxinus</i>
<i>Hyacinthus</i>	<i>Delphinium</i>	<i>Pelargonium</i>	<i>Strychnos</i>
<i>Allium</i>	<i>Papaver</i>	<i>Tropaeolum</i>	<i>Nerium</i>
<i>Lilium</i>	<i>Brassica</i>	<i>Citrus</i>	<i>Tectona</i>
<i>Iris</i>	<i>Raphanus</i>	<i>Cedrela</i>	<i>Verbena</i>
<i>Ixia</i>	<i>Cheiranthus</i>	<i>Manihot</i>	<i>Capsicum</i>
<i>Gladiolus</i>	<i>Matthiola</i>	<i>Mangifera</i>	<i>Solanum</i>
<i>Musa</i>	<i>Amelanchier</i>	<i>Euonymus</i>	<i>Lycopersicum</i>
<i>Zingiber</i>	<i>Sorbus</i>	<i>Vitis</i>	<i>Nicotiana</i>
<i>Dendrobium</i>	<i>Eryobotrya</i>	<i>Gossypium</i>	<i>Physalis</i>
<i>Cattleya</i>	<i>Pyrus</i>	<i>Malva</i>	<i>Petunia</i>
<i>Oncidium</i>	<i>Cydonia</i>	<i>Sterculia</i>	<i>Datura</i>
<i>Odontoglossum</i>	<i>Prunus</i>	<i>Elodea</i>	<i>Calceolaria</i>
<i>Cypripedium</i>	<i>Rubus</i>	<i>Begonia</i>	<i>Sesamum</i>
<i>Phalaenopsis</i>	<i>Crataegus</i>	<i>Opuntia</i>	<i>Pavetta</i>
<i>Vanilla</i>	<i>Fragaria</i>	<i>Eucalyptus</i>	<i>Psycotria</i>
<i>Salix</i>	<i>Rosa</i>	<i>Oenothera</i>	<i>Benincasa</i>
<i>Populus</i>	<i>Heteromeles</i>	<i>Aralia</i>	<i>Cucumis</i>
<i>Juglans</i>	<i>Dolichos</i>	<i>Hedera</i>	<i>Cucurbita</i>
<i>Castanea</i>	<i>Lathyrus</i>	<i>Carota</i>	<i>Citrullus</i>
<i>Corylus</i>	<i>Indigofera</i>	<i>Pastinaca</i>	<i>Sicyos</i>
<i>Morus</i>	<i>Kraunhia</i> (?)	<i>Levisticum</i>	<i>Echinocystis</i>
<i>Pouzolzia</i>	<i>Lupinus</i>	<i>Apium</i>	<i>Ageratum</i>
<i>Cannabis</i>	<i>Mucuna</i>	<i>Arbutus</i>	<i>Chrysanthemum</i>
<i>Acalypha</i>	<i>Phaseolus</i>	<i>Vaccinium</i>	<i>Lactuca</i>
<i>Humulus</i>	<i>Vigna</i>	<i>Ardisia</i>	<i>Blumea</i>
<i>Ficus</i>	<i>Pisum</i>	<i>Crispandisia</i>	<i>Synedrella</i>
<i>Rheum</i>	<i>Trifolium</i>	<i>Amblyanthus</i>	<i>Tragopogon</i>
<i>Polygonum</i>	<i>Medicago</i>	<i>Amblyanthopsis</i>	<i>Bellis</i>
<i>Atriplex</i>	<i>Arachis</i>	<i>Diospyros</i>	<i>Aster</i>
<i>Spinacia</i>	<i>Acacia</i>	<i>Ligustrum</i>	

PERIOD OF GREATEST SUSCEPTIBILITY

In certain diseases the brief seedling stage of the plant is the one most subject to attack, e. g., Stewart's disease of maize due to *Bacterium Stewarti*, and brown rot of tomato and tobacco due to *Bacterium Solanacearum*, but many bacterial diseases of older plants are also rather strictly time-limited. In both groups it is a question of abundant immature tissue. To the latter class belong the numerous leaf-spots, fruit-spots, and blights, e. g., black spot on the plum and peach, due to *Bacterium Pruni*, and fire-blight of the pear, apple, quince, etc., due to *Bacillus amylovorus*. In such cases, so far at least as they occur in temperate climates, the disease appears in

the spring and the greater part of it occurs during a brief period in the early summer, in which growth of roots, leaves and shoots is proceeding rapidly and there are many young and succulent parts. The cause of the disease may and often does remain on the plant over winter in a latent or semi-latent condition (walnut blight, pear blight, plum canker), but the active period is limited to three months, more or less, of actively growing weather in which developing tissues, subject to infection, are abundant. With definitive growth and the hardening of the tissues in late summer and autumn, the disease is checked and disappears, or remains as a slow canker to appear again on other parts the following spring. It is a very instructive experiment to see, for example, inoculations of *Bacillus amylovorus* on ripening fruits and shoots of the pear wholly fail toward the end of July, which were eminently successful on the same trees at the beginning of June. The difference in this case is not due to lessened virulence on the part of the organism, but to changes in the host-plant, making it non-susceptible. Similar changes leading to non-susceptibility occur in the Japanese plum subject to *Bacterium Pruni*; the young fruits are very susceptible, the maturing fruits cannot be infected.

Other parasites on the contrary are able to attack, disintegrate and destroy matured tissues, e. g., the pith of cabbage stems, turnip roots, the ripened tubers of the potato, well developed roots of sugar beets, the bulbs of onions and hyacinths, full-grown melon and cucumber fruits.

In both of these types the action of the parasite is expended chiefly on the parenchyma, although in some cases (the plum disease, Appel's potato rot) there is more or less bacterial invasion of the local vessels. Vascular occupation is not a special characteristic.

In the typical vascular diseases the case is reversed. Here parenchyma is also destroyed, more or less, but the most conspicuous and destructive action is on the vascular bundles themselves, which are occupied for long distances, to the death, or great detriment, of the whole plant. In maize attacked by *Bacterium Stewarti*, it is not unusual, indeed one might rather

say it is customary, to find the vessels of the stem filled with the bacteria continuously for a distance of 3-6 feet from the point of infection, i. e., from the surface of the earth to the top of the full-grown plant. In cucurbits attacked by *Bacillus tracheiphilus* and in sugar-cane attacked by *Bacterium vascularum* the same thing occurs, and many of the vessels are filled solid with the bacterial slime to a distance of 8 or 10 feet from the place of infection. In such cases infection has taken place generally near the base of the plant, which continues to grow for some weeks or months.

Transitions, of course, occur. *Bacterium Stewarti*, for example, is confined much more strictly to the vascular bundles of the maize stem than is *Bacterium Solanacearum* to those of the tomato, potato, or tobacco stem, although it also is a vascular parasite; that is, following infection of the vessels we do not find in the maize stems that extensive breaking down of the pith and phloem into vast cavities which is so common, for example, in tobacco and tomato stems.

WHAT GOVERNS INFECTION

Within the plant we may suppose, from certain indications, that abundant juiciness is the chief factor governing the infection of immature tissues. To this may be added an abundant well-adapted food supply and, in some cases, probably the absence of inhibiting substances, which may appear later. As the parts approach maturity the water content becomes less. Along with this, acids, sugars, amids, proteids, etc., are consumed and converted into substances less well adapted to the needs of the meristem-parasites, if not wholly inimical. In young shoots of potato and tomato, or of pear and apple, as contrasted with old ones, or in the roots of carrots as compared with the leaves, or in rapidly-growing cabbages, as compared with slow-growing ones, we know that there is an excess of water, and this alone appears to be sufficient to explain the difference in behavior of their respective parasites in old versus young parts. When, however, we come to ripening fruits, such as the pear and the plum, it would seem that they are still juicy enough to favor the growth of almost any

bacterium, and we are forced to the hypothesis of chemical changes within the fruits to account for the failure of inoculations. As a rule (there are striking exceptions), parasitic micro-organisms are rather sensitive to changes in their environment, e. g., to drying, exhaustion of food supplies, multiplication of their own by-products, conversion of an easily assimilable substance into one less assimilable or actually harmful, appearance of esters, new acids, etc. But why speculate! Much additional experimenting must be undertaken before we shall have precise and full data. We are still largely in the observational stage.

The parasites of ripened tissues do not require so much water, are able to convert starch into sugar, or have a special liking for some other element of the plant tissue.

Externally, a number of factors favor infection. One of these is excessive shade, either of clouds or of foliage. Another is high temperature. When these two factors are accompanied by excessive rainfall, wet earth, and heavy dews, the conditions are ideal for the rapid dissemination and the destructive prevalence of a variety of bacterial diseases of cultivated plants. The bean spot due to *Bacterium Phaseoli*, the black spot of plum due to *Bacterium Pruni*, and the larkspur disease due to *Bacterium Delphinii*, are all favored by heavy dews and by shade. In hot, wet weather in July pear blight due to *Bacillus amylovorus* often bursts out like a conflagration and sweeps over whole orchards. In warm, moist autumns bacterial diseases of the potato may destroy almost or quite the entire crop over extensive districts.

HOW INFECTION OCCURS

As I have already described elsewhere how infection occurs,¹ I will only dwell for a moment on it here, offering a few examples.

The commonest way of infection is probably through wounds.

¹ Smith, E. F. Bacteria in relation to plant diseases. Carnegie Inst. Washington, Publ. 27^a: pp. 51-64. 1911.

In Italy, the olive tubercle due to *Bacterium Savastanoi* has been observed to begin very often in wounds made by hail-stones. In South Africa, crown-gall is said to be disseminated in the same way. In this country and also in Sumatra, *Bacterium Solanacearum* enters the plant more often than otherwise through broken roots. A tomato or tobacco plant with unbroken roots will thrive in a soil deadly to one that has been root-pruned. I have myself observed this. We may suppose that substances attractive to the particular bacteria diffuse into the soil from the broken roots, following which they enter the plant. Resistant plants may be supposed to diffuse indifferent or repellant substances. All infections must be chemotactic.

More interesting perhaps are those diseases which begin in natural openings, i. e., in places where the protective covering of the plant gives place to special organs such as nectaries, water-pores, and stomata.

All the pome fruits subject to fire-blight are liable to blossom infection. The bacteria multiply first in the nectaries of the flower, passing down into the stem by way of the ovary and pedicel. Blossom blight of the pear is a very conspicuous and common form of the disease as everybody knows. Thousands of blighted blossom clusters may be seen in any large orchard subject to this disease.

In the black rot of the cabbage due to *Bacterium campestre*, the majority of the infections begin in the water-pores. These are grouped on the margins of the leaf at the tips of the serratures. From this point the bacteria burrow into the vascular system of the leaf and so pass downward into the stem and upward into other leaves.

In the black spot of the plum, almost or quite all of the infections are stomatal. A large proportion of them are also stomatal in the leaf-spot of cotton, and other leaf-spots.

TIME BETWEEN INFECTION AND APPEARANCE OF THE DISEASE

As in animal diseases, the period of latency may be very short or surprisingly long. Some time must be allowed the parasitic organism to multiply inside the plant before it does

damage serious enough to be recognized externally as a *disease*. This is the so-called "period of incubation," during which the parasite is growing and its enzymes and toxins are becoming active. The microscope shows it to be present in the tissues, but the latter have yielded only a little in the immediate vicinity of the bacterial focus. This time is short or long depending on whether the parasite or the host has the first advantage. If the host is growing rapidly it may either entirely outstrip the parasite, or be only so much the more subject to it. All depends on whether the parasite finds the initial conditions entirely suited to its needs, or by means of its secretions and excretions can quickly make them so, and consequently can from the start make a rapid growth, or must first slowly overcome obstacles of various sorts, such as inhibiting acids and resistant tissues. The plant may show signs of infection within as short a time as one or two days after inoculation (various soft rots), or it may be as long a time as one to two months before they appear (Cobb's disease of sugar-cane, Stewart's disease of sweet-corn). In the latter, infection generally occurs in the seedling stage and the maize plant may be three months old and six feet tall before it finally succumbs. Of course, as in case of bacterial animal diseases, the greater the volume of infectious material the shorter the time. I have seen many instances of that law. In general, the period of latency may be said to vary from one to three weeks (yellow disease of hyacinth, black rot of cabbage, black spot of plum, cucurbit wilt, pear blight, angular leaf-spot of cotton, sorghum leaf-stripe, etc.).

RECOVERY FROM DISEASE

Mention has already been made of the self-limited spot diseases and blights. As the actively growing season draws to a close such diseases cease their activity.

Also in some plants well developed signs of vascular disease may be suppressed (squash, maize, sugar-cane) or remain in abeyance for a longer or shorter period, according to the varying fortunes of the host and the capabilities of the parasite. The tomato plants inoculated with *Bacterium Sol-*

anacearum (Medan III) and photographed for Volume III of 'Bacteria in Relation to Plant Diseases' (plate 45 D), entirely outgrew the disease, as did also certain sugar-canes (series VI) inoculated with *Bacterium vascularum*.¹ Also, I have seen tomato plants recover only to develop a second and fatal attack of the vascular brown rot three months after the first attack, during which period they had made an extensive healthy-looking growth.²

Recovery from disease may depend on *loss of virulence* on the part of the parasite. This often occurs when bacteria are grown for some time on culture-media, and it occurs also in nature, but its cause is obscure.

AGENTS OF TRANSMISSION

These may be organic or inorganic. In many cases the plant itself harbors the parasite indefinitely, carrying it over from year to year on some portion of its growth.

Seeds, tubers, bulbs, grafts, or the whole plant may be responsible for the appearance of the disease the following year in the old localities, and through the agency of seedsmen, nurserymen, or whoever disseminates plants, for outbreaks in regions hitherto exempt.

There is good reason to believe that the black rot of cabbage and Stewart's disease of sweet corn have been disseminated broadcast in the United States in recent years by ignorant and unscrupulous seedsmen. Both diseases are transmitted to seedling plants from the seed. The yellow disease of hyacinths is carried in the bulb. Potato tubers from diseased fields may infect healthy fields. Apple grafts have transmitted crown-gall. Slightly infected trunks and limbs of trees (hold-over pear blight, walnut blight, canker of the plum) may infect shoots, leaves, blossoms, or fruits the following season. The soil around the infected plant may serve for years as a source of infection to other species (crown-gall), or to other individuals of the same kind (various leaf-spots). Occasionally, however, a parasite seems to die out of certain soils (*Bac-*

¹ Smith, E. F. Bacteria in relation to plant diseases. Carnegie Inst. Washington, Publ. 27³: p. 33. 1914.

² *Ibid.* p. 179.

terium Solanacearum). The pear blight organism probably dies out of soils quickly as it does in a majority of the blighted branches. Pear blight by soil infection is not known.

Among extraneous agents, wind and water have been suspected. I have never seen any clear indications of wind-borne infection, not even when conditions seemed to invite it, but water often carries parasites and furnishes conditions favorable to infection. Horne has shown that the olive tubercle in California is transmitted in this way. Honing, in the tobacco fields of Sumatra, has traced infection several times to the watering of plants from infected wells, and has cultivated the parasite from the water. I have discovered experimentally that to obtain several sorts of bacterial leaf-spots (bean, cotton, peach, plum, carnation, larkspur, sorghum, geranium) the surface of the leaves must be kept moist to the same extent they would be in case of prolonged dews or frequent light showers. Such conditions are necessary to enable the bacteria to penetrate the stomata and begin to grow. In case of water-pores, however, the plant itself furnishes the water necessary for infection, if the nights are cool enough, i. e., if the air remains near enough to saturation to prevent for some hours the evaporation of the excreted water from the leaf-serratures. Every plant with functioning water-pores awaits its appropriate bacterial parasite. The genus *Impatiens* is a good example. I have looked on it for one in vain but I am sure it must occur.

Man and the domestic animals, especially through the agency of the dung-heap, infallible repository of all sorts of discarded refuse, undoubtedly help to spread certain bacterial diseases of plants (potato rots, black rot of cabbage, etc.).

Birds probably transmit some of these diseases on their feet or in other ways. In connection with the bud-rot of the coconut palm in the West Indies, I suspect the turkey-buzzard, but the evidence is not complete. Long since, Mr. Waite obtained (once in Florida, once in Maryland) the strongest kind of circumstantial evidence going to show that pear blight may be spread by birds.

Respecting insects, molluscs, and worms, the evidence is complete. They often serve to carry these diseases. I have summarized our knowledge in another place¹ and will here content myself with a brief statement calling renewed attention to the subject.

We had very good evidence of the transmission of one bacterial disease of plants by insects long before the animal pathologists awoke to the importance of the subject,² but it cannot be said that they have ever paid much attention to it, although it antedates by two years the work by Theobald Smith and Kilborne showing that Texas fever is transmitted by the cattle tick (*Ixodes bovis*). That discovery also belongs to the credit of the U. S. Department of Agriculture, and the two together may be said to have laid broad and deep the foundations of this most important branch of modern pathology. Waite isolated the pear blight organism, grew it in pure cultures, and proved its infectious nature by inoculations. With such proved cultures he sprayed clusters of pear flowers in places where the disease did not occur and obtained blossom-blight, and later saw this give rise to the blight of the supporting branch, found the organism multiplying in the nectar, and reisolated it from the blighting blossoms. On some trees he restricted the disease to the sprayed flowers by covering them with mosquito netting to keep away bees and other nectar-sipping insects. On other trees where the flowers were not covered he saw bees visit them, sip from the inoculated blossoms and afterwards visit blossoms on unsprayed parts of the tree which then blighted. Finally he captured bees that had visited such infected blossoms, excised their mouth parts, and from these, on agar-poured plates, obtained *Bacillus amylovorus*, with colonies of which he again produced the disease. These experiments were done in several widely separated localities with identical results. I saw them and they made a great impression on me.

¹ Smith, E. F. Bacteria in relation to plant diseases. Carnegie Inst. Washington, Publ. 27²: p. 40. 1911.

² Waite, M. B. Results from recent investigations in pear blight. Bot. Gaz. 16: 259; Am. Assoc. Adv. Sci., Proc. 40: 315. 1891.

The writer has since proved several diseases to be transmitted by insects, notably the wilt of cucurbits, and here the transmission is not purely accidental, but there appears to be an adaptation, the striped beetle (*Diabrotica vittata*), chiefly responsible for the spread of the disease, being fonder of the diseased parts of the plant than of the healthy parts. This acquired taste, for it must be that, works great harm to melons, squashes, and cucumbers. Whether the organism winters over in the beetles, as I suspect, remains to be determined. Certainly the disease appears in bitten places on the leaves very soon after the spring advent of the beetles.

In 1897 I showed that molluscs sometimes transmit brown rot of the cabbage, and last year I saw indications in Southern France which lead me to think that snails are responsible for the spread of the oleander tubercle, i. e., I saw them eating both sound and tubercular leaves, and found young tubercles developing in the eroded margins of bitten leaves.

Parasitic nematodes break the root tissues and open the way for the entrance of *Bacterium Solanacearum* into tobacco and tomato, as was first observed by Hunger in Java and later by myself in the United States. One of the serious problems of plant pathology is how to control *Heterodera radiculicola*, not only because of its wide distribution on a great variety of cultivated plants and the direct injury it works, but also on account of the often very much greater injury it causes through the introduction into the roots of the plant of bacterial and fungous parasites. The man who shall discover an effective remedy will deserve a monument more enduring than brass. Our Southern States in particular are overrun with this parasite.

Much remains to be done before we shall know to what extent fungous parasites function as carriers of parasitic bacteria. H. Marshall Ward sought to explain the presence of bacteria in diseased plants by supposing that they must enter the plant through the lumen of fungous hyphae. In this he was wrong, certainly if it be stated as a general proposition, but it appears to be clear that in some cases the two types of parasites work together, the fungus invading first, and the

bacterium following hard after and often doing the major part of the damage. The reverse of this also occurs, the bacterium entering first and the fungus following.

Parasitic bacteria are soon followed by saprophytic bacteria which complete the destruction of the tissues, and, if the disease is somewhat advanced, cultures from the tissues may yield only the latter (potato rots). Also, as in animals, one parasitic disease may follow another and the second be more destructive than the first, e. g., fire-blight following crown-gall on the apple.

EXTRA-VEGETAL HABITAT OF THE PARASITES

Here is perhaps the place to say a few words about the non-parasitic life of the attacking organisms.

All are able to grow saprophytically, i. e., on culture media of one sort or another, and probably all live or may live for a time in the soil. Very few, however, have been cultivated from it. The vast mixture of organisms present in a good earth rather discourages search. In some of the unsuccessful attempts failure may have been due to not having undertaken isolations at exactly the right time, or in just the right place, or on just the proper medium, but more often probably to the swamping tendency of rapidly growing saprophytes. How long a parasite is able to maintain its virulent life in a soil must depend largely on the kind of competitors it finds. I have used the term *virulent*, because it is conceivable that an organism might remain alive in a soil long after losing all power to infect plants, just as we know it can in culture media. *Bacterium Solanacearum* causing brown rot of *Solanaceae*, *Bacillus phytophthorus* causing basal stem rot and tuber rot of the potato, and *Bacterium tumefaciens* causing crown-gall, certainly live in the soil, and the soundest plants when set in such soils, especially if wounded, are liable to contract the disease, if they belong to susceptible species. The root-nodule organism of *Leguminosae*, which I have not considered here, also lives in many soils, as every one knows.

MORPHOLOGY AND CULTURAL CHARACTERS OF THE PARASITES

Most of the plant bacteria are small or medium sized rod-shaped organisms. Very few parasitic coccus forms are known. In fact, none are very well established. Some of these bacteria are Gram positive, others are not. All take stains, especially the basic anilin dyes, but not all stain with the same dye or equally well. Most of the species are motile by means of flagella—polar or peritrichiate. A few are non-motile, genus *Aplanobacter*.¹ Some develop conspicuous capsules, others do not. Few, if any, produce endospores. Grown pure on culture media in mass, they are either yellow, pure white, or brownish or greenish from the liberation of pigments. Red or purple parasites are not known. We formerly supposed that there were no green fluorescent species capable of parasitism, but now several are known, e. g., the organism causing the lilac blight of Holland, with pure cultures of which the writer obtained typical infections at Amsterdam in 1906, and afterwards in the United States (now first recorded). Some species produce gas, liquefy gelatin, consume asparagin, destroy starch, and reduce nitrates; others do not. Their fondness for sugars and alcohols is quite variable. Some are extremely sensitive to sunlight and dry air (*Bacillus carotovorus*, *Bacillus tracheiphilus*). Others are remarkably resistant, remaining alive and infectious on dry seeds for a year (*Bacterium campestre*, *Bacterium Stewarti*, *Aplanobacter Rathayi*). Some are strictly aerobic, others can grow in the absence of air, if proper foods are available. Some are very sensitive to acids, alkalies and sodium chlorid, others are not. Some have wide ranges of growth from 0°C. upwards. Some will not grow at or near 0°C., others will grow at or above 40°C. Very few, however, will grow at blood temperature, certain ones even in plants or on culture media are killed by summer temperatures, and none are known definitely to be animal parasites.

¹ Smith, E. F. Bacteria in relation to plant diseases. Carnegie Inst. Washington. Publ. 27¹: p. 171. 1905; *Ibid.* 27²: pp. 155, 161. 1914.

ACTION OF THE PARASITE ON THE PLANT

In some cases it is hard to draw the line between parasitism and symbiosis or mutualism. Probably we shall find more and more of these transition states. I have included *Ardisia* in my list of genera and have excluded the genera of legumes subject only to root nodules. But a nodule on the root of a legume, so far as the local condition is concerned, is a disease as much as a leaf-spot, and, if Nobbe and Hiltner's statements are to be credited, the general effect of the root-nodule organism on the plant may be excessive and injurious and not to be distinguished from a disease.¹

In the tropical East Indian *Ardisia*, which is one of the strangest cases of mutualism known to me, and on which Miehe has done such a beautiful piece of work, we perhaps have something akin to what occurs in the root nodules of legumes. Here the bacterial injury is local and internal. There are no superficial indications of disease. The bacteria are most abundant in the leaf-teeth where they form pockets or cavities and multiply enough to make the leaf serratures appear blanched or yellowish and slightly swollen, but never enough to kill them. In smaller numbers the bacteria occur in other parts of the plant including the inner parts of the seed from which they are transmitted to the seedling, whose leaf serratures, infected through their water-pores, in turn become the chief focus of the bacterial multiplication. Apparently the bacteria are always present, and we do not know what would happen to *Ardisia* plants grown without them, nor do we know how to obtain such plants. It would be an interesting experiment to see if they could be produced and to watch their behavior.

The action of such organisms as I have mentioned differs probably from the behavior of active parasites in that they liberate much weaker toxins and enzymes, can attack only very actively growing parts, and also give off compensating nitrogenous substances. Not yet proved for *Ardisia*.

¹ Smith, E. F. Bacteria in relation to plant diseases. Carnegie Inst. Washington, Publ. 27²: p. 131, last paragraph. 1911.

The active parasites produce toxins freely, poisoning the tissues, and enzymes converting starches into sugars, complex sugars into simpler ones, and so on, for their nutrition. They also neutralize and consume plant acids, and feed upon amido bodies and other nitrogenous elements of the host. As a result of their growth, many of them liberate both acids and alkalis to the detriment of the plant. The solvent action of their products on the middle lamellae separates cells and leads to the production of cavities in the bark, pith, phloem and xylem. There is also, or may be, a mechanical splitting, tearing or crushing due to the enormous multiplication of the bacteria within confined spaces. The whole intercellular mechanism may be honeycombed and flooded in this way, and if the cavities are near the surface the tissues may be lifted up or the bacteria may be forced to the surface through stomata in the form of tiny beads or threads (pear, plum, bean, maize, sugar-cane, etc.), or by a splitting process. The splitting in the black spot of plum fruits and peach fruits, however, results from local death of the attacked tissue with continued growth of the surrounding uninjured parts.

A majority of the forms known to cause plant diseases are extra-cellular parasites occupying chiefly the vessels and intercellular spaces, causing vascular diseases, soft rots, spot diseases, etc. But intra-cellular parasites also occur, e. g., *Bacterium Leguminosarum* causing root-nodules on legumes, and *Bacterium tumefaciens* causing crown-gall. The former multiplies within the cell myriadfold, prevents its division, destroys its contents including the nucleus, and enormously stretches the cell wall so that the cell becomes much larger than its normal fellow cells and is packed full of the bacteria. The latter does not multiply abundantly within the cell, does not enlarge it, does not injure its viability, and would be a harmless messmate were it not for the fact that it exerts a stimulating effect on the cell nucleus, compelling the cell to divide again and again.

THE REACTION OF THE PLANT

We now come to the reaction of the plant. What response does it make to this rude invasion? Ten years ago we might

have said, "With rare exceptions, the plant is passive or nearly so," but that would have been a superficial observation.

In every disease we must suppose that the plant makes some effort to throw off the intruder, although often its forces are paralyzed and overcome very early in the progress of the disease.

One of the most conspicuous results is lessened growth. In some of my plants recovering from brown rot due to *Bacterium Solanacearum*,¹ a month after external signs of the disease had disappeared the check plants were twice the size of the inoculated ones, and there was still a very decided difference after more than two months. I do not know how to explain this checked growth unless it be the response to absorbed toxins.

On potato plants attacked early by *Bacterium Solanacearum* the tubers remain small. On maize attacked by *Bacterium Stewarti* the ears are imperfect. Olive shoots inoculated and infected by *Bacterium Savastanoi* are always dwarfed, and the crown-gall dwarfings are frequently very conspicuous. The dwarfing of melon and squash plants attacked by *Bacillus tracheiphilus* is also conspicuous. Uninoculated sugar-cane stems soon surpass in height and vigor those successfully inoculated with *Bacterium vascularum*.

Changes in color are also conspicuous. The attacked parts may become greener than normal, or fade to yellow, red, brown or black. In tomato fruits there is often a retarded ripening on the attacked side with persistence of the chlorophyll. Crown-galls on daisy are greenish. In certain leaf-spots also the leaf green persists in the vicinity of the spot while the rest of the leaf becomes yellow (bean-leaf spot). The male inflorescence of maize attacked by *Bacterium Stewarti* ripens prematurely and becomes white.

Distortions of various kinds appear (leaves of bean, lilac, larkspur, hyacinth, mulberry, Persian walnut). The leaves of tomato plants attacked by *Bacterium Solanacearum* are bent downwards; so are the fronds of the coconut palm when

¹ Smith, E. F. Bacteria in relation to plant diseases. Carnegie Inst. Washington, Publ. 27^a: pl. 45-D. 1914.

attacked by the bacterial bud-rot. Knee-shaped curvatures of the culms appear on *Dactylis* attacked by *Aplanobacter Rathayi*, and in the buds of the sugar-cane attacked by Cobb's disease.

Organs may be developed in excessive number or out of place, as roots in hairy-root of the apple, witch-brooms on *Pinus*, and incipient roots on the stems of tomato, tobacco, chrysanthemum, nasturtium, etc. Hunger found a bud on a tomato leaflet which he attributed to the stimulus of *Bacterium Solanacearum*.

In various diseases the plant removes starch from the vicinity of the bacterial focus which it endeavors to wall off by the formation of a cork barrier, and in this effort it is sometimes successful if the parasite is growing slowly.

The most conspicuous response of the plant is in the form of pathological overgrowths,—cankers, tubercles, and tumors. Some of these are very striking, e. g., those on the ash, olive, pine, oleander, and on a multitude of plants attacked by crown-gall. In some of these growths there is a great reduction of the vascular system, and a great multiplication and simplification of the parenchyma. There are also various other phenomena nearly related to what takes place in certain insect galls. In crown gall cell division under compulsion proceeds at such an abnormally rapid rate that the cells are forced to divide while still immature, and in this way masses of small-celled unripe (anaplastic) tissue arise. These develop tumor-strands on which secondary tumors arise.

PREVALENCE AND GEOGRAPHICAL DISTRIBUTION

Economically considered, bacterial diseases of plants may be classed as major or minor. Most of the leaf-spots would fall into the latter class. Various soft rots, blights and vascular diseases, being wide-spread and destructive to plants of great economic importance, may be classed as major diseases. Cankers and tumors would fall midway in such a grouping. Occasionally a minor disease, e. g., lettuce rot, celery rot, under favorable conditions may assume great importance.

It will be of interest to mention a few of these diseases with particular reference to their distribution and prevalence.

Dutch East Indies.—The tobacco disease of Sumatra and Java is probably the most destructive, if the Sereh of sugar-cane is not bacterial. Each of these diseases has caused enormous losses. Each threatens an industry. The tobacco disease occurs also in the West Indies, in the United States, and probably also in South Africa. If Janse's root disease of *Erythrina*, the coffee shade tree of Java, is also bacterial, as he supposed, then there is another great bacterial plague in that region, for hundreds of thousands of trees have died, and another species has been substituted as a shade tree.

West Indies.—Here the most destructive disease is the bacterial bud-rot of the coconut palm, which occurs all around the Caribbean, and threatens the entire destruction of a profitable industry in Cuba. There is also the bacterial disease of bananas and plantains, but the most wide-spread and destructive *Musa* disease of the Western Hemisphere is the Panama disease, due to a *Fusarium*.

Australia.—Cobb's disease of sugar-cane has probably attracted more attention in Australia than any other bacterial trouble, although bacterial rots of the potato are also very destructive. The cane disease in both Queensland and New South Wales has in many cases destroyed the output of whole plantations and greatly discouraged planters. This disease occurs also in Fiji, and probably in South America.

Japan.—Probably the tobacco wilt, which has destroyed many fields, is the worst Japanese disease. This is believed to be identical with the tobacco wilt of Sumatra and of the United States. Several other bacterial blights have been reported, including one of the basket willow.

India.—The brown rot of *Solanaceae* is common and destructive. Most of Asia is a *terra incognita*.

South Africa.—The mango disease in recent years has greatly reduced the exports. Potato and tomato wilts are common. There is a serious tobacco disease, probably bacterial. Crown-gall is common and injurious on shade and orchard trees. Other diseases occur.

South America.—There is a serious disease of sugar-cane in Brazil and another in Argentina, both of which I believe are of bacterial origin, and identical with Cobb's disease. Bondar has reported a destructive manihot disease. The bud-rot of the coconut occurs in the north.

United States and Canada.—Potato rots probably cause the greatest losses one year with another. Following these I should think pear and apple blight. Perhaps the latter should be placed first, for the destruction of an acre of potatoes would scarcely equal the value of a single fine pear tree, and thousands are destroyed every year. In California, which was free from pear blight until recently, the losses in the last fifteen years have been enormous, amounting to about one-third of all the full-grown orchards and to a money-loss estimated at \$10,000,000 for the five years preceding the efforts for its restriction begun in 1905 by the U. S. Department of Agriculture. Very serious losses from this disease are experienced every year in the East, or were until growers became generally familiar with methods of control.

In our southern states the tobacco and the tomato wilt have made it impossible to grow these crops on many fields. In the northern United States the cucurbit wilt is wide-spread and destructive, but cucurbits are of course a minor crop.

The walnut blight has done much damage in California. This occurs also in New Zealand and Tasmania.

The bacterial disease of alfalfa has been serious in parts of the West. It is most injurious early in the season, i. e., on the first cutting.

Holland.—Here the yellow disease of hyacinths is always destructive and will eventually put an end to hyacinth-growing for export if means cannot be had for its control, since the land suited for hyacinths is limited in amount. Brown rot of cabbage occurs in Holland and Denmark, and is common now also in many parts of the United States. It was probably imported into the United States from Denmark on cabbage seed. Some years in nurseries about Amsterdam the lilac blight has been troublesome.

Great Britain and Germany.—Potato rots are probably the most destructive bacterial diseases.

France and Italy.—Potato diseases are common. Olive tubercle, common also in California, and all around the Mediterranean, is prevalent in spots. Vine diseases, especially *Maladie d'Oleran* and crown-gall, do considerable damage. Pear blight seems to be absent in France, but has been reported from several places in Italy. The destructive Italian rice disease, *brusone*, is not due to bacteria as reported, but to a fungus (*Piricularia*).

METHODS OF CONTROL

In conclusion, some words on prophylaxis will be in order. Until recently almost nothing was known. Unfortunately so far as regards most of these diseases, methods of control must still be worked out. But with rapidly increasing knowledge of the biological peculiarities of the parasites causing these diseases, and of the ways in which they are disseminated, light begins to dawn, so that before many years have passed we may confidently expect the more intelligent part of the public to be applying sound rules for the control of these diseases,—rules based on the individual peculiarities of the parasites and carefully worked out experimentally by the plant pathologist.

The little that we now know may be summarized in part as follows:

Waite has shown that pear blight winters over in exceptional trees on trunk and limbs in the form of patches which ooze living bacteria the following spring and are visited by bees and other insects, and that if these "hold-over" spots are cut out thoroughly over regions several miles in diameter (wide as a bee flies), the disease does not appear on the blossoms and shoots the following spring, except as it is introduced into the margins of this area from remoter uncontrolled districts. He has tried this method of control very successfully, both in Georgia and California. Sometimes only one tree in many carries over the disease, but such is not always the case, and the success of this method involves the inspection of every pome tree in a district with complete eradication

of every case of the hold-over blight, and this in great fruit regions requires a small army of trained inspectors. During the blighting period in late spring and early summer, if one would save his orchard, the trees must be cut over for removal of diseased material as often as every week, and in the worst weather oftener.

The introduction of diseases transmitted by way of seeds, bulbs, and tubers may be avoided by obtaining these from plants not subject to the disease. As this freedom cannot always be known, bulbs and tubers should be inspected critically before planting, and firm-coated seeds should be soaked for 15 minutes in 1:1000 mercuric chlorid water. In case of two plants (cabbage and maize) we know positively that the diseases are transmitted on the seed and this is probably true for several others—beans, sorghum, orchard grass. All shrivelled seeds should be screened out before planting.

The seed bed in case of tobacco, tomato, cabbage, and transplanted plants generally, should be made on steam-heated or fire-heated soil, or new earth which one has good reason to think free from the parasite in question.

Nematode-infected soil should be avoided.

Cuttings of carnations, chrysanthemums, roses, peaches, plums, apples, quinces, sugar-cane, etc., used for slips, buds, or grafts should be from sound plants. By following this practice, recommended in case of sugar-cane by Cobb, the more intelligent cane planters in New South Wales have overcome the disease due to *Bacterium vascularum*.

On badly infested soils a careful long rotation should be practised and the low places should be drained.

Certain diseases may be held in check by germicidal sprays. Pierce reduced the number of infections in walnut blight fifty per cent by this method. Scott and Rorer combated leaf-spot of the peach in this way, the sprayed trees retaining their leaves, the unsprayed ones becoming defoliated. ——— in Italy has recommended it and used it successfully on olive trees following hail-storms to keep out the olive tubercle.

When diseases are transmitted by insects the destruction of the latter must receive prompt attention.

Great care should be taken to keep the manure heap free from infection. Diseased rubbish should be burned or buried deeply. It must not be thrown into a water supply or fed to stock or dumped into the barnyard.

It has been found that some varieties of plants are less subject to disease than others (pear, apple, plum, maize, potato, tomato, sugar-cane, banana, cabbage, etc.), and there are also individual variations within the variety. These phenomena lead us to hope that by selection, or hybridization, valuable resistant strains may be originated. Meanwhile the resistant sorts when they are of any value commercially should be substituted for sensitive sorts in localities much subject to the disease. Unfortunately some of the resistant sorts have other less desirable qualities. A vast amount of experimental work must be done in this field before we shall have substantial results, and at least a generation or two will be required to learn even the boundaries of the field. But the problem offered is so enticing and has such immediately practical bearings that in the near future we may suppose many pathologists will devote themselves to it, and that long before the whole field is worked over, many useful results will be forthcoming. The labor involved is enormous and exacting to discouragement at times, the results come so slowly, so much must be done to be certain of so little, all because the organisms dealt with are very small—*how small*, we seldom realize!

Many a time in the past when downcast I have repeated to myself Seneca's rolling words, *Palma non sine pulvere per viam rectam*, and have had more or less encouragement out of them. They are a good motto for any man, since nothing is more certain than this, that without plenty of well-directed hard work there can be no worthy success in any field of human endeavor.